Interruption of Behavior, Inescapable Shock, and Experimental Neurosis: A Neurophysiologic Analysis*

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Abstract—Experiments reported in the literature involving interruption of behavior and leading to symptoms of anxiety in conditioned rats are analyzed from the neurophysiologic point of view and compared with the effects of inescapable electric shocks in dogs. It is suggested that anxiety and neurotic behavior (failure to attempt to escape from shock) are accompanied by a state of high ergotropic excitation which leads to a "spilling over" of ergotropic discharges into the trophotropic system. The resulting simultaneous ergotropic and trophotropic discharges impinging on the cerebral cortex are thought to induce abnormal emotions and behavior. Normal behavior is restored by reducing excessive ergotropic activity and re-establishing reciprocal relations between the ergotropic and trophotropic systems.

In 1965 I presented a theory of anxiety based on a physiologic analysis of various forms of experimental and clinical neuroses in terms of the activities of, and the mutual relations existing between, the trophotropic and ergotropic systems (Gellhorn, 1965; Gellhorn and Loofbourrow, 1963). In the meantime the work of Mandler *et al.* (Mandler, 1964; Mandler and Watson, 1966), who emphasized that anxiety is produced through interruption of behavior, came to my attention. These investigations pose two questions:

1. Is the interpretation of anxiety presented in my earlier study applicable to Mandler's experiments and kindred observations?

2. What is the physiologic mechanism associated with the appearance of anxiety following interruption of behavior and neurosis-producing conditions?

The Nature of Anxiety

PAVLOV, by his work on the conditional reflex method of measuring excitation and inhibition through salivary secretion, reported in 1911 and in 1923, opened up the field for the study of abnormal psychopathological states in the dog. It was his idea that "experimental neurosis" was caused by a "collision" between excitation and inhibition in the brain. In 1925 he postulated the importance of the "type" of nervous system in determining nervous breakdown (Pavlov, 1929).

Since then extensive studies have shown that in the normal animal a positive conditional stimulus (CS) elicits ergotropic and a

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negative CS trophotropic symptoms, the former consisting of sympathetic discharges, increased muscle tone, and desynchronization of cortical potentials, and the latter comprising parasympathetic discharges, relaxation of skeletal muscles, and cortical synchrony (Gellhorn, 1967). The ergotropic and trophotropic excitations are more intensive and extensive in the neurotic state. Moreover, the state of anxiety as seen in the neurotic organism is accompanied by the simultaneous or nearly simultaneous appearance of trophotropic and ergotropic discharges, whereas in the normal organism the trophotropic and ergotropic systems show reciprocal relations (Gellhorn, 1967). Study of the EEG supports this interpretation. In the neurotic state delta potentials of low frequency and high amplitude occur together with beta waves of high frequency and low amplitude, seemingly due to nearly-simultaneous activation of the ergotropic and trophotropic systems. In addition, sleeplike conditions in which the tone of the skeletal muscles is greatly increased ("pseudodecerebrate" rigidity, Anderson and Parmenter, 1941) may appear. These and other data previously discussed by this author (1965, 1967) suggest that neurosis-producing stimuli evoke an excitation which spreads from the ergotropic to the trophotropic system or heightens the excitability of both systems to such a degree that on rather mild stimulation, or "spontaneously," both systems discharge.

This hypothesis is supported by the following findings:

1. Procedures such as massive pain and repeated insulin-induced hypoglycemic comas which activate the ergotropic *and* the trophotropic systems produce anxiety neurosis (Rose, Tainton-Pottberg and Anderson, 1938).

2. The most common method of evoking a neurotic condition is the application of an unconditional stimulus (US) which acts primarily on the ergotropic system (such as shock or an air blast) while the trophotropic system is in a state of heightened activity as, for instance, during eating (Masserman, 1943).

3. As the signs of anxiety (fear of the food box and various forms of displacement activities) in a preneurotic animal disappear, excessive trophotropic discharges are lessened and reciprocal relations between the trophotropic and ergotropic systems are restored (Jacobsen and Skaarup, 1955).

Study of the psychogalvanic reflex in states of phobias in man discloses a related phenomenon: not only a positive but also a negative (nonreinforced) CS evokes this reflex, whereas in the normal subject only the positive CS is effective (Alexander, 1961). This contribution to ergotropic excitation of stimuli which normally

enhance trophotropic excitation is reduced or abolished when, through drugs (such as meprobamate) and other procedures, the excitability of the ergotropic system is reduced. These physiologic changes are accompanied by clinical improvement.

Anxiety as the Result of Interruption of Behavior

Mandler's (1964) first study on rats was carried out with hungry animals which had learned brightness discrimination in a Y-maze with large pellets of food serving as US. When these animals were subjected to extinction trials in a *satiated* state they showed "extremely excited behavior" consisting of jerky and convulsive movements. These symptoms occurred regularly in the satiated rats and increased in intensity in successive trials. They occurred rarely in the animals which had been deprived of food for 24 hours as in the preceding learning experiments. Mandler suggested that this disorganized behavior was due to two factors:

1. The interruption of an organized sequence which led to the ingestion of food;

2. The state of satiation which deprived the animals "of any other situationally-relevant behavior."

The conclusion that distress is related to the "unavailability of alternative responses," particularly in man, was confirmed in further experiments.

Closely related to these observations are the results of an experiment of Mowrer and Viek (1948) which I quote from Mandler's paper (1966): "Two groups of rats were trained to approach a certain place to feed. Following mastery of this task, both groups were shocked at the place where they had been fed. One group had available an instrumental response, jumping, which terminated the shock, but the other group of animals had no such instrumental response available to turn off the shock. The second group was yoked to the first, however, so that the amount and duration of the shock was identical for the two groups. Mowrer and Viek found that the group which had an instrumental response available showed much less disruption of the previously learned approach behavior than did the group which had no escape response available."

The behavioral consequences of inescapable shock were further investigated in dogs tested in escape-avoidance situations (Overmier and Seligman, 1967). Conditional escape responses to shock in a shuttle-box were delayed and reduced in frequency after the dogs had been exposed to inescapable shock in a hammock. Moreover, dogs which had learned to panel-press in order to escape shock showed normal escape-avoidance behavior in a shuttle box, whereas

"yoked, inescapable shock in the harness produced profound interference with subsequent escape responding in the shuttle box" (Seligman and Maier, 1967). After a few trials in the shuttle box these dogs seemed "to give up" and did not jump at all in response to the shocks. Other experiments dealt with the question of whether retroactive procedures would have a curative effect on the action of inescapable shock. This was indeed the case: repeated pulling of the dog to the safe side of the shuttle box during the application of the conditional and unconditional stimuli restored escape and avoidance behavior (Seligman, Maier and Geer, 1967). Since these experiments show that neurotic behavior may be produced by inescapable shocks *per se,* they are, at least in part, responsible for the behavioral disturbances seen in Mowrer and Viek's work.

The studies of inescapable shock and the experiments of Richter (1957) showed that anxiety and abnormal behavior result from exposure of animals to situations which do not offer alternate responses, although it is questionable whether the experimental conditions involved interruption of behavior.

Neurophysiologic Analysis

It may be assumed that the animals in Mandler's experiments were in a state of high ergotropic excitability at the end of the period of conditioning, and that this state persisted for some time after the extinction trials were begun. Under these circumstances the behaviors of food-deprived and satiated rats were significantly different: anxiety indicated by autonomic discharges and convulsive movements appeared on extinction in the satiated group but not in the food-deprived group. Physiologically it has been shown that the lateral hypothalamus controls feeding and the ventromedial hypothalamic nuclei regulate satiety. The former is part of the ergotropic and the latter part of the trophotropic system.

At the beginning of the extinction experiment the satiated animals seem to be in a state in which strong ergotropic *and* trophotropic discharges occur at the hypothalamic level, whereas the food-deprived rats are subjected only to an increased ergotropic activity characteristic of the state of conditioning. Results of this experiment are therefore, in agreement with the results of the work referred to in the introduction (Gellhorn, 1965, 1967), demonstrating that conditions which tend to precipitate strong ergotropic and trophotropic discharges simultaneously lead to a variety of neurotic symptoms which characterize anxiety.

A different interpretation of this experimental situation should not be overlooked. Since satiation or stimulation of the ventromedial nucleus of the hypothalamus inhibits the feeding center, reduces

emotional reactivity (Wheatley, 1944) and enhances cortical synchronization (Clemente, Sterman, and Wyrwicka, 1964), one might expect that satiation would reduce the activity of the ergotropic system. This is undoubtedly true at relatively low ranges of ergotropic activity,* but in states of high ergotropic activity tending to show the "spilling over" of ergotropic discharges into the trophotropic system this effect must be magnified when these discharges summate with the enhanced activity of the trophotropic system induced by satiation.

A different mechanism seems to account for the finding by Mowrer and Viek (1948) that anxiety reactions were more frequent in conditioned animals which could not escape a shock than in those which terminated it by jumping although the degree of nociceptive stimulation was the same in both groups. It is assumed that the vigorous movement seen in the group which by jumping controlled the electric shock, in some manner had an influence on the intensity of the emotional responsiveness. This brings to mind an experiment of Freeman and Pathman (1942) who studied the effect of movements of the whole body on the restitution of emotions which had been aroused by strong sensory stimuli (pistol shot) or conflictual situations. The palmar conduction increased under these conditions, but the return of this ergotropic (sympathetic) reaction to control levels was accelerated when the emotional excitement was associated with gross movements. Apparently, the duration of the emotional reaction indicated by the psychogalvanic reflex was lessened when it was accompanied by a marked motor response.

In the light of more recent research, the following phenomena seem to account for this important finding. Movements enhance, through proprioceptive feedback on the hypothalamic system (Gellhorn, 1958, 1964), the intensity and abruptness of the ergotropic discharge which has been induced by the emotion. Such discharges have been shown to be followed by equally abrupt trophotropic discharges. Thus, stimulation of the posterior hypothalamus resulting in a rise in blood pressure and acceleration of the heart rate is followed by a fall in blood pressure and a sudden slowing of the heart rate, the degree of which parallels the intensity of the preceding sympathetic excitation (Gellhorn, 1959). Since there is in general a parallelism between the intensity of the peripheral and central (corticipetal) discharges of the ergotropic and trophotropic systems, it may be assumed that this brief "rebound" shifts the balance to the trophotropic side. Under these circumstances the activity of the anterior hypothalamus is increased and

* See Gellhorn (1957, 1960, 1967) for a detailed discussion of the state of trophotropic (parasympathetic) tuning.

inhibits the cortical and peripheral discharges originating in the posterior hypothalamus. Consequently, palmar conduction is restored quickly and the muscles relax. It seems probable that this mechanism is likewise involved in restitution of the emotional balance following crying and in the relaxation after convulsive laughter and subsequent to the strong ergotropic discharges of orgasm.

A similar trophotropic rebound following rapid ergotropic discharges, indicated by jumping in response to an electric shock (Mowrer and Viek and Seligman *et al.*), reduces ergotropic excitation and emotional excitement, whereas in the absence of this homeostatic mechanism in "helpless" animals the central ergotropic discharges continue to increase (afterdischarges) and to produce anxiety.

Several data support this interpretation and its applicability to animals subjected to inescapable shock:

(1) Liddell (1956) states that inescapable shock leads to neurosis;

(2) dogs subjected to this procedure in the work of Seligman *et al.* show symptoms resembling the inhibitory form of experimental neurosis;

(3) massive parasympathetic discharges have been observed in this condition (see Overmier and Seligman, 1967);

(4) passive transfer of these dogs to the safe side of the shuttle box re-establishes escape and avoidance behavior. This therapeutic effect is related to the observation of Kopa *et al.* (1962) that the environment in which a shock has been applied repeatedly, induces (in this writer's interpretation) a state of enhanced ergotropic reactivity (ergotropic tuning). In contrast to this effect a "safe" environment increases trophotropic responsiveness. In these dogs the curative effects of pulling them over to the safe side and, also, the presence of the investigator are based on the shift of the ergotropic-trophotropic balance to the trophotropic side: Gantt (1962) mentions that the mere presence of a person reduces the heart rate of a neurotic dog from 140–180 to 60–70/minute and inhibits "the marked respiratory changes usually evoked by an anxiety-producing stimulus."

The fact that in states of greatly heightened activity of the ergotropic system the excitation "spills over" into the trophotropic system is further illustrated in Richter's (1957) experiments on wild rats forced to swim in conditions in which neither a rest (through floating) nor an escape was possible. If the whiskers were trimmed the rats died under these conditions in a few minutes. This was probably due to the increased emotional excitation in a new situation in which sensory exploration of the environment was impaired, but

even normal rats died after relatively brief periods. The heart rate first accelerated but declined markedly later. At death there was a standstill of the heart in diastole. Cholinergic drugs aggravated and atropine retarded these effects. These data and the finding of gross hypothalamic lesions at autopsy suggest that in this condition intensive trophotropic discharges were associated with states of strong ergotropic excitation and that the hypothalamic system is primarily involved.

Conclusion

The importance for the organism of carrying out a plan has been stressed by Miller, Galanter and Pribram (1960); interference with the plan has been thought to be the cause of distress and anxiety (Freud, 1935; Lewin, 1936). This problem has been reinvestigated in man and animals by Mandler and Watson (1966), who suggest that "a situation which interrupts or threatens the interruption of organized response sequences, and which does not offer alternate responses to the organism, will be anxiety-producing." However, a formulation of the underlying mechanisms whereby "interruption leads to a state of arousal which is followed by emotional behavior" (Mandler, 1964) has been lacking. The explanation presented in this paper is supposed to fill this gap and was shown to be applicable to behavioral disturbances resulting from inescapable shock.

Our analysis shows that a single basic physiologic mechanism underlies the great variety of emotional and neurotic symptoms which have been studied experimentally: it involves a high degree of excitation of the ergotropic system which "spills over" into the trophotropic system, thereby interfering with the reciprocal relations which prevail between the two systems under physiologic conditions. Factors which facilitate this process induce anxiety and neurosis; procedures which diminish ergotropic discharges, eliminate excessive trophotropic activity, and thereby restore ergotropic–trophotropic balance and reciprocal behavior between the two systems tend to restore normal behavior. The prevention of anxiety in experiments permitting alternate responses seems to be based on a homeostatic rebound reaction of the trophotropic system which follows movements carried out during a high degree of ergotropic excitation.

On this basis it is suggested that the abnormal behavior is due at least in part to the fact that the cerebral cortex is subjected to intensive discharges from the ergotropic and trophotropic systems at the same time. It is not improbable that this mechanism is involved in the loss of performance at high degrees of ergotropic activity also. Whereas the heart rate increases with increasing degrees of water deprivation, a graph of the frequency of bar-

pressing for water takes the form of an inverted U under these conditions (Belanger and Feldman, 1962), and a similar effect is seen in man when performance is measured under increasing degrees of tension (Cohen, Silverman and Burch, 1956; Wood and Hokanson, 1965; Malmo, 1967).

The following data and considerations should be borne in mind:

1. The monotonic reaction of the heart rate and the curvilinear relation of performance to increasing degrees of stress suggest that different levels of the nervous system are activated in the two processes and that performance depends on a higher level than changes in heart rate;

2. Metabolism and sensitivity to anoxia, hypoglycemia, and the action of certain drugs are greatest in the phylogenetically-young parts of the brain (Himwich, 1951). These data may account for the fact that injection of meprobamate has a differential effect on the two functions: the changes in heart rate remain unaltered but the decrement in bar-passing for food is abolished in 30-60 hour food-deprived animals (Malmo and Belanger, 1967). It is assumed, therefore, that in instrumental conditioning experiments the high degrees of ergotropic excitation, associated with prolonged food or water deprivation and similar stresses, leads to simultaneous ergotropic and trophotropic discharges which act rather selectively on those brain structures involved in performance.

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